

# THE OPTIMAL RATIO OF FAT-TO-CARBOHYDRATE IN THE DIET<sup>1</sup>

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## ABSTRACT

A major issue in human nutrition is the optimal relation of carbohydrate-to-fat in the diet. According to some investigators, a high proportion of fat energy to total energy favors the development of several chronic diseases. Among these are obesity, coronary heart disease, diabetes, and cancer. The theory that a high proportion of fat relative to other nutrients promotes the development of obesity is founded on research with experimental animals and in human population surveys. This theory has been difficult to prove in prospective feeding studies in humans; therefore it remains a contentious issue. Regarding coronary heart disease, little evidence supports a claim that a high proportion of dietary fat predisposes to disease. On the other hand, strong evidence bolsters the claim that certain fatty acids raise the risk for coronary heart disease. These include saturated fatty acids and *trans* fatty acids, both of which raise serum cholesterol levels. In contrast, neither monounsaturated nor polyunsaturated fatty acids raise serum cholesterol levels and seemingly pose little risk for coronary disease. The relationship between dietary fat and type 2 diabetes is tied largely to the issue of obesity, because obesity is a major cause of diabetes. Although animal studies and epidemiological studies have implicated dietary fat as a factor in cancer, recent prospective epidemiological data in humans have cast doubt on the possibility of a strong relationship. In summary, clear evidence points to the need to reduce intakes of saturated and *trans* fatty acids in the diet. Beyond this change, a balanced ratio of unsaturated

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fatty acids to carbohydrate leading to fat intake of approximately 30% of total energy seems appropriate for the American public.

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## CONTENTS

INTRODUCTION .....	326
MAJOR ISSUES FOR MACRONUTRIENT RECOMMENDATIONS .....	327
<i>Macronutrients and Chronic Disease</i> .....	327
<i>Specific Diseases at Issue</i> .....	327
<i>Individual Versus Population Recommendations</i> .....	327
<i>Criteria for Evidence</i> .....	329
<i>Practical Considerations</i> .....	329
<i>Fat/Carbohydrate Ratios</i> .....	329
<i>Fat Composition</i> .....	330
MACRONUTRIENT RATIOS AND THE DEVELOPMENT OF CHRONIC DISEASE .....	331
<i>Obesity</i> .....	331
<i>Atherogenesis</i> .....	332
<i>Thrombogenesis</i> .....	335
<i>Insulin Responsiveness and Type 2 Diabetes</i> .....	335
<i>Carcinogenesis</i> .....	336
<i>Osteoporosis</i> .....	336
MACRONUTRIENT RECOMMENDATIONS .....	337
<i>Dietary Fat Composition</i> .....	337
<i>Dietary Carbohydrate Composition</i> .....	338
<i>Percentage of Dietary Fat</i> .....	338

## INTRODUCTION

A highly contentious dispute surrounds the optimal ratio of carbohydrate-to-fat in the diet. One view holds that excess fat in the diet contributes to several chronic diseases. This position favors the recommendation for a low-fat (high-carbohydrate) diet. Another opinion contends that higher intakes of fat, particularly certain types of fat, protect against these diseases. Because these differences in view are held by reputable scientists, the issue of the optimal fat/carbohydrate ratio obviously is confounded. The uncertainty that clouds these two viewpoints does not detract from the importance of the issue. Both fats and carbohydrates are major constituents of the diet; a successful resolution of the dispute would have a major impact on eating habits of the general population. Even though there are large vested interests favoring one or another pattern of diet composition, a solid determination of the optimal intake of dietary fat would eventually lead to a change in the public's eating habits. This review outlines the major considerations that surround the fat-carbohydrate debate. Much of the discussion is couched in terms of dietary fat; it must be remembered, nonetheless, that modifications in dietary fat intake usually imply

a reciprocal change in carbohydrate intake. Finally, the issue goes beyond the fat/carbohydrate ratio; any discussion of dietary fat requires a consideration of its heterogeneity in fatty acid composition.

## MAJOR ISSUES FOR MACRONUTRIENT RECOMMENDATIONS

### *Macronutrients and Chronic Disease*

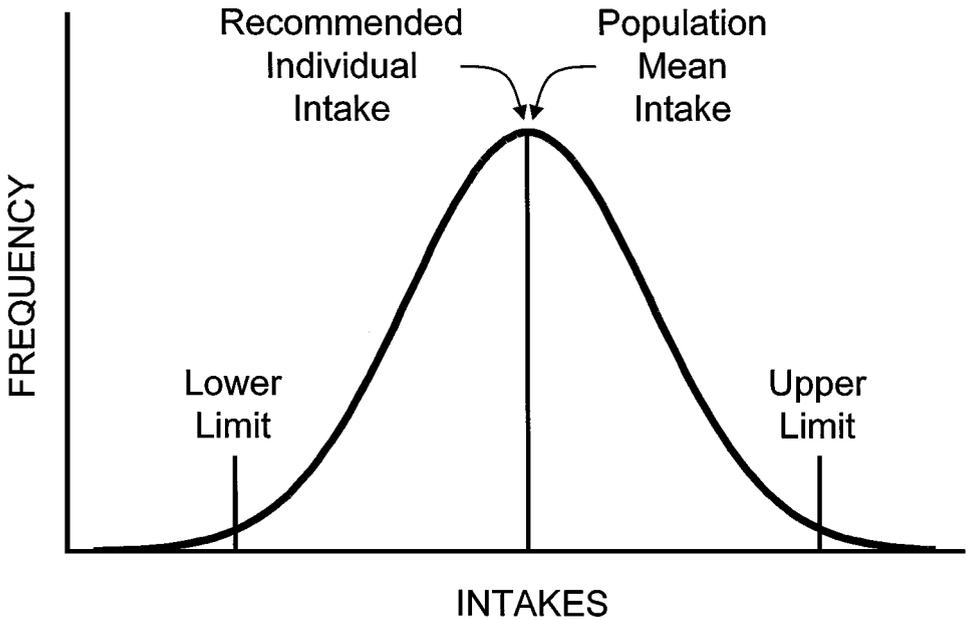
The human body is equipped to effectively utilize both carbohydrate and fat. In addition, both nutrients are required for normal metabolic processes. The body cannot function exclusively on carbohydrate and protein; for this reason, if the body is deprived of dietary fat, fatty acids will be synthesized to meet its metabolic or structural needs. On the other hand, the theory has been put forth that long-term ingestion of relatively high amounts of fat will engender small metabolic imbalances that predispose individuals to chronic disease. This theory stands at the heart of the macronutrient controversy. Without question, high intakes of either fat or carbohydrate are well tolerated over the short run. The central question is whether long-term ingestion of larger quantities of either will produce small detrimental effects that accumulate over time and predispose individuals to various chronic diseases. This question is the focus of this review.

### *Specific Diseases at Issue*

The major diseases that have been linked to chronic macronutrient imbalances include coronary heart disease (CHD), stroke, cancer, diabetes mellitus, and osteoporosis. Each disease thus represents an endpoint in the creation of dietary fat recommendations. Among these, CHD stands at the fore. As societies become more urbanized and affluent, CHD emerges as the number one killer. Because intakes of fat typically increase during urbanization of societies, excess fat becomes particularly suspect. Furthermore, high intakes of dietary fat have been implicated in the causation of stroke, cancer, and diabetes; these latter conditions, therefore, are discussed as well.

### *Individual Versus Population Recommendations*

The field of dietary recommendations in general has long been confounded by ambiguities as to whether individuals or populations are the principal target of recommendations (4). This confusion extends to macronutrient recommendations. Presumably, most recommendations from authoritative bodies are meant to apply to individuals, and the consequences of these recommendations for populations generally have not been taken into account. Recommendations for



*Figure 1* Critical cut points for population intakes for specific nutrients. The extremes of safety (or practicality) are represented by the *lower limit* and *upper limit*. The population mean intake is that recommended to optimally reduce risk of a disease in a population. For most dietary fat recommendations, the recommended population mean intake corresponds closely to the recommended individual intake. This correspondence may not hold for other nutrients.

populations nonetheless are important for the broad planning of food production and delivery.

The relationship between recommendations for individuals and populations, as they might apply to dietary macronutrients, are shown in Figure 1. These relationships are particularly applicable to the percentage of dietary energy in the form of fat or fatty acids. The essential cut points on the curve of population intakes are the upper limit, lower limit, recommended individual intake, and recommended population mean intake (4). The upper limit basically represents the upper level of safe intakes. For macronutrients, the lower limit relates as much to practicality as to nutritional requirement. The recommended intake for individuals is an approximate “optimal” intake that balances safety, benefit, and practicality. The recommended population mean intake is set between upper and lower limits; for the whole population, it usually is close to the recommended individual intake. For particular populations, however, recommended intakes for individuals and populations may diverge (4).

### *Criteria for Evidence*

Decisions pertaining to appropriate and/or optimal intakes for individuals and populations should be based on scientific evidence. This evidence is of several types: biochemical studies, investigations in animal models, epidemiological surveys, clinical investigation (and human feeding studies), and clinical trials (4). All of these except the latter provide evidence of association; they do not prove that modification of the diet will prevent disease. Ultimate proof of causality can come only through controlled clinical trials with humans. However, in the field of nutrition, controlled clinical trials typically are expensive and are difficult to perform. Consequently, recommendations often must be based on an authoritative judgment that evidence of other types is congruent. Judgments of experts currently underlie most macronutrient recommendations. Indeed, many of the unresolved issues about optimal intakes of fat and carbohydrate will probably never be resolved through controlled clinical trials; instead, recommendations will be made largely on the basis of accumulated evidence of other types.

Because of the paucity of controlled clinical trials to bolster macronutrient recommendations, epidemiological data acquire increased importance in making judgments about optimal macronutrient ratios (39, 41). Various populations differ greatly in amounts and types of fat ingested, and they also differ in incidence and prevalence of chronic disease (28). It is tempting to draw causal inferences from these differences. Still, most populations vary in other respects as well, including diet composition and other life habits. Thus, causal inferences that attempt to link macronutrient composition to disease must be made with the recognition that the probability of confounding variables is considerable.

### *Practical Considerations*

Various populations also differ in availability of food supplies, traditional cultural habits, economic resources, and preoccupation with other life issues. Translation of scientific evidence into population recommendations must take these other factors into account. Unless this is done, recommendations will fall on deaf ears.

### *Fat/Carbohydrate Ratios*

Differences in food supply in different areas of the world have led to marked regional differences in ratios of fat-to-carbohydrate in the diet; this ratio typically is expressed in terms of percentage of total dietary energy derived from fat (28). In Asia, for example, most nutrient energy comes from carbohydrates; traditionally, fat has contributed only 10%–20% of total energy. At this level of intake, the diet can be called a very-low-fat diet. In contrast, in the Mediterranean basin, the percentage of dietary fat can reach 40% of total energy (a high-fat

diet); in this region, most of the fat derives from plant sources, particularly olive oil. In northern Europe, animal fats traditionally have been consumed in large quantities, and high-fat diets also have been the rule; even so, the composition of dietary fat differs markedly from that of southern Europe. Finally, in some populations, fat intakes are intermediate, averaging about 30% of total energy (a moderately low-fat diet). There is a common belief among nutritionists that high-fat diets per se foster a predisposition to chronic disease, whereas very-low-fat (very-high-carbohydrate) diets are protective. The scientific basis of this hypothesis must be examined carefully before it can be accepted.

A consideration of total dietary fat intakes cannot be divorced from dietary carbohydrate. The percentage of energy provided by protein varies little; thus, the percentage of carbohydrate must increase as percentage of fat declines. Moreover, high-carbohydrate intakes may not be entirely neutral with respect to chronic disease when high intakes are ingested for prolonged periods. Like fat, carbohydrate elicits unique metabolic reactions, and prolonged ingestion of high-carbohydrate diets theoretically could have detrimental effects.

### *Fat Composition*

Dietary fat, which consists largely of triacylglycerol, can be highly heterogeneous. The fatty acid pattern of dietary triacylglycerol is shown in Table 1. Each

**Table 1** Dietary fatty acids<sup>a</sup>

Fatty acid	Symbol <sup>b</sup>
Saturated	
Palmitic	C <sub>16:0</sub>
Myristic	C <sub>14:0</sub>
Lauric	C <sub>12:0</sub>
Caproic	C <sub>10:0</sub>
Caprilic	C <sub>8:0</sub>
Stearic	C <sub>18:0</sub>
<i>Trans</i> -mono	<i>Trans</i> C <sub>18:1,n-9</sub>
Unsaturated	
<i>Cis</i> -monounsaturated	<i>Cis</i> C <sub>18:1,n-9</sub>
Polyunsaturated	
n-6	C <sub>18:2,n-6</sub>
n-3	DHA, EPA

<sup>a</sup>The letter n and number locates number of carbon atoms from terminal methyl group where first double bond appears. DHA, docosahexanoic acid (C<sub>22:6,n-3</sub>); EPA, eicosapentanoic acid (C<sub>20:5,n-3</sub>).

<sup>b</sup>First number denotes number of carbon atoms; second number denotes number of double bonds.

fatty acid has unique features in its metabolism, and each potentially could have long-term effects, either beneficial or detrimental. Any recommendations about dietary fat should take its heterogeneity into account. To date, research has only scratched the surface in its attempt to understand the long-term metabolic effects of different fatty acids.

Below, these general issues are discussed first in the light of the relationship between dietary fat and common chronic diseases. Consideration then is given to long-term effects of dietary carbohydrate because a discussion of total dietary fat cannot be divorced from carbohydrate. Moreover, an effort is made to synthesize what is known about the metabolism of each fatty acid as it relates to chronic disease.

## MACRONUTRIENT RATIOS AND THE DEVELOPMENT OF CHRONIC DISEASE

### *Obesity*

One argument against the consumption of a high-fat diet is that it may predispose individuals to obesity. Without doubt, a high intake of total fat can contribute excess energy and thus can promote or support obesity. The critical question is whether a high percentage of dietary fat promotes weight gain more than a low percentage of fat intake. Because fat is higher in caloric density than carbohydrate, it has been claimed that consumption of a high-fat diet will almost inevitably be accompanied by a higher energy intake than will occur on a low-fat (or very-low-fat) diet (44). According to one hypothesis, a high intake of fat stimulates the appetite and induces overconsumption of food (active hyperphagia) (12). According to a second, excessive weight gain results from passive hyperphagia, i.e. increased energy intake resulting unawares from the high caloric density of fat-enriched foods (44). A third hypothesis is that some animals have a sluggish capacity to oxidize fatty acids (34); if so, a high percentage of fat in the diet will lead to accumulation of excess fat in the body. Perhaps the strongest support for each of these theories comes from studies of animals. Among these, passive hyperphagia seems to explain most of the weight gain observed in animals fed high-fat diets (51). Some studies (12, 34) with animals, however, lend support to the other theories. These various studies of laboratory animals have led many investigators to conclude that high-fat diets are an important cause of obesity in animals; if so, they might also predispose humans to obesity.

A limited amount of epidemiological data from humans appears to support the notion that high-fat diets promote development of obesity. Among populations that differ in eating habits, those with a higher consumption of total fat usually have a higher prevalence of obesity (48). Feeding studies in humans, on the other hand, reveal that high-fat diets per se do not cause weight gain when

compared isocalorically with very-low-fat diets (20). Thus, if high-fat diets promote obesity in free-living populations, the mechanism must be fat-induced active hyperphagia (12), or overconsumption of energy unawares (passive hyperphagia) (44). Defects in fatty acid oxidation resulting in accumulation of body fat apparently are rare.

To date, no controlled clinical trials have demonstrated that modifying the percentage of fat appreciably affects body weight in free-living populations (48a). The few reported studies have provided ambiguous results; furthermore, increments in body weight following long-term ingestion of high-fat diets generally have been small. Controlled clinical trials to test this question are difficult to carry out. Once participants enter a clinical trial, they are no longer free living. Moreover, any weight gain that occurs secondary to a high-fat diet can be overridden by a conscious reduction in energy intake. To test the theory of passive hyperphagia, conscious factors must be eliminated from the study, with only unconscious factors being allowed to play. This presumably occurs in experiments with animals. For humans, however, conscious control of food intake becomes an important factor; almost certainly a conscious reduction in food intake can prevent weight gain on high-fat diets. This fact is well illustrated in many populations in which a sizable portion of the population maintains a desirable content of total body fat in spite of consuming a high-fat diet.

Of course, it could be argued that a subgroup of the population is susceptible to developing obesity when consuming high-fat diets. This argument, however, is brought into question by the recent weight gain that has occurred broadly in the United States population simultaneously with a decrease in percentage fat in the diet (6). This weight gain likely is the result of an increased consumption of high-carbohydrate foods. Presumably other factors predominate over the passive hyperphagia accompanying high-fat diets in the causation of obesity among Americans (53). Certainly obesity is a multifactorial disease, being related both to a high-energy intake and a reduced energy expenditure (16). Many factors besides the fat content of the diet affect both energy intake and expenditure and thus modify body weight. Consequently, it is difficult to define precisely the contribution of passive hyperphagia from high-fat diets to the prevalence of obesity. In all likelihood, the high prevalence of obesity in the United States can be explained in part by excessive consumption of total energy; but the extent to which excessive energy intake results passively from a high percentage of fat in the diet is not known (53). Even more uncertain is whether a recommendation for a low-fat diet will reduce the prevalence of obesity.

### *Atherogenesis*

Early investigations in laboratory animals engendered the diet-heart hypothesis, namely the concept that much of CHD results from dietary excesses.

Certainly the finding that dietary cholesterol causes atherosclerosis in rabbits and other animals was a strong impetus for this hypothesis (49). Even so, investigations using humans revealed that high intakes of cholesterol do not produce the marked hypercholesterolemia observed in many laboratory animals (26); in other words, humans are relatively resistant to excess dietary cholesterol, although excess intakes do produce mild-to-moderate increases in serum cholesterol levels (17). In the 1950s, the focus of the diet-heart hypothesis was changed to animal fats in general. Investigations at this time revealed that diets high in animal fats produce higher cholesterol levels than those rich in vegetable fats (1, 25, 29). Follow-up research rapidly implicated saturated fatty acids as the culprit for raising serum cholesterol, i.e. they are cholesterol-raising fatty acids (1, 19, 25, 27). In contrast, unsaturated fatty acids, particularly polyunsaturated fatty acids, appeared to be cholesterol-lowering fatty acids (1, 21, 25, 27). As a result of these studies, the diet-heart hypothesis came to target saturated fatty acids as the major dietary cause of atherosclerosis.

Subsequent investigations of several types supported this hypothesis. In animal and human feeding studies, saturated fatty acids consistently raise serum cholesterol levels when compared with unsaturated fatty acids (18). Also, populations consuming high intakes of saturated fatty acids have higher rates of CHD than do those consuming lesser amounts (28). These findings prompted attempts to develop controlled clinical trials to test whether reducing saturated fatty acids, and replacing them with unsaturated fatty acids, will reduce risk for CHD. Several small trials (9, 11, 21, 31, 50) suggested the benefit of risk reduction from this exchange. Still, in the 1970s, influential investigators concluded a major diet-heart trial was not feasible, because of high costs and impracticality (40). Nonetheless, on the basis of other lines of evidence, there is a widespread acceptance of the concept that diets high in saturated fatty acids, which definitely raise serum cholesterol levels, foster a predisposition to CHD when consumed over a period of many years (39, 41).

In spite of the general acceptance of the atherogenic properties of saturated fatty acids, an area of continuing dispute concerns the desirable replacement of saturated fatty acids in the diet. Early clinical investigation focused on linoleic acid because it appeared to induce a greater lowering of serum cholesterol levels than alternative nutrients, e.g. oleic acid and carbohydrate (19, 27). For this reason, several of the earlier clinical trials (9, 11, 21, 31, 50) compared diets high in saturates with those high in linoleic acid. The suggestive benefit from this exchange, observed in these trials, increased enthusiasm for linoleic acid as a replacement for saturates. On the other hand, no large populations have ever consumed large amounts of linoleic acid for prolonged periods. This latter fact raised concern in the minds of many because the long-term safety of high intakes of polyunsaturates was not assured by population data. In contrast,

in the Mediterranean region, where olive oil represents the major source of fat, the diet contains large amounts of the monounsaturated oleic acid, and CHD incidence is low (28); moreover, no evidence of harm from high intakes of olive oil exists. These epidemiological findings have led other researchers to favor replacement of saturated fatty acids with oleic acid. Recent reports (33, 37) that oleic acid, when substituted for saturated fatty acids, lowers low-density lipoprotein (LDL)-cholesterol levels almost as much as linoleic acid has heightened enthusiasm for this replacement.

An alternative approach to dietary reduction of LDL-cholesterol has been engendered by the fact that very-low-fat (high-carbohydrate) diets have traditionally been consumed in Asia, and in this region, rates of CHD are low (28). Other populations consuming very-low-fat diets likewise have low rates of CHD. These epidemiological findings have encouraged many nutritionists to favor very-low fat diets over high intakes of unsaturated fatty acids to reduce the risk for CHD. This position is bolstered by the belief that very-low-fat diets curtail the development of obesity and may reduce the risk for other diseases. Metabolic studies (15, 35) revealed that very-low-fat diets reduce serum LDL-cholesterol levels as much as do those high in unsaturated oils; thus, from the standpoint of LDL-cholesterol levels, the two approaches are equivalent. On the other hand, the effects of a diet high in unsaturates and one high in carbohydrates on all lipoprotein species are not equivalent. For example, a diet very low in fat and high in carbohydrate modifies several lipoprotein species in the direction of an abnormal pattern called the atherogenic lipoprotein phenotype (10, 15, 35). This phenotype is commonly observed in patients with CHD; it is characterized by elevated triglycerides, small LDL particles, and low high-density lipoprotein-cholesterol levels (3). Each of these changes has been implicated in atherogenesis. In addition, even though LDL-cholesterol levels are similar on diets high in either carbohydrates or monounsaturated fatty acids, the smaller LDL particles accompanying high-carbohydrate diets are indicative of higher levels of LDL-apolipoprotein B (10). Furthermore, on a high-carbohydrate diet, apolipoprotein B levels in tricylglycerol-rich lipoproteins also are higher. Although some investigators dismiss the significance of the atherogenic lipoprotein phenotype occurring on a high-carbohydrate intake, at the very least the question must be raised whether this dietary pattern is entirely innocuous for long-term atherogenesis.

The sources of dietary carbohydrate are an issue of some potential importance. Digestible forms of carbohydrate include simple sugars and starches. Indigestible forms of carbohydrate go under the name of fiber. The term complex carbohydrate is commonly employed as a category different from simple sugars, but this term is confusing, as both starch and fiber are complex carbohydrates.

There is a general impression among some investigators that starch has fewer adverse metabolic consequences than simple sugars. For example, it has been reported that simple sugars are more likely to produce hypertriglyceridemia than starch (32). This is true in certain experimental animals, although it has not been proven unequivocally in humans. Some forms of fiber, e.g. soluble fiber, appear to slow absorption of carbohydrate and thus produce an attenuated glucose response (42); ingestion of sizable quantities of soluble fiber also leads to a small reduction in LDL-cholesterol levels (13). These effects of fiber, however, must be distinguished from those of starches. Metabolic differences resulting from ingestion of predominant simple sugar versus starch have not been adequately explored.

Although most attention has been directed toward the relationship between macronutrients and lipoproteins, the diet could affect atherogenesis in other ways. Limited data in experimental animals raise the possibility that certain fatty acids could promote atherogenesis independently of lipoproteins (30). Unfortunately, this possibility has never been fully examined in either animals or humans. Limited data also suggest that the dietary fat content or fatty acid composition modulates the blood pressure (23); if so, this too could affect the rate of atherogenesis. Currently, however, we simply do not know whether changes in macronutrient composition affect atherogenesis independently of changes in serum lipoproteins.

### *Thrombogenesis*

It has been claimed that diets high in fat induce a prothrombotic state. For example, high-fat diets may raise factor VII activities (38). Studies in laboratory animals have further implicated saturated fatty acids in thrombogenesis; stearic acid in particular has been singled out as being prothrombotic (8). Epidemiological studies further suggest that saturated fatty acids as a group foster a predisposition to thrombosis (46). However, in spite of these sparse reports, there is not a consensus view that high-fat diets contribute to venous thrombosis, coronary thrombosis, or thrombotic strokes. Much more research is required to confirm or refute a clinically significant effect.

### *Insulin Responsiveness and Type 2 Diabetes*

Because insulin secretion by pancreatic  $\beta$ -cell is glucose sensitive, a high percentage intake of carbohydrate should produce higher postprandial insulin levels. This response in fact has been reported (5, 45). Moreover, it is possible that repeated stimulation of a high insulin output by high-carbohydrate diets could hasten an age-related decline in insulin secretion and lead to an earlier onset of type 2 diabetes. Of interest, the severity of postprandial glycemia and insulin

response may depend on the quality as well as the quantity of carbohydrate ingested. Ingestion of simple sugars could evoke a greater excursion of plasma glucose and insulin than do starch-rich foods.

It has been reported that high-carbohydrate diets, compared with higher intakes of monounsaturates, enhance resistance to the peripheral action of insulin (43), but this claim was not confirmed by another study (16). Thus, the long-term consequences of high-fat versus high-carbohydrate diets on peripheral insulin sensitivity remain to be adequately defined.

### *Carcinogenesis*

Many investigators have long held the view that high-fat diets predispose to cancer (2, 7). This view has been based in part on research in animals. High-fat diets, particularly those rich in polyunsaturated fatty acids, can promote carcinogenesis in some experimental animals (47). A link between dietary fat and cancer, moreover, has been suggested in epidemiological studies, particularly in migrating populations (2, 7). Several studies (2, 7) indicate that when people emigrate from a region where low-fat diets are consumed to regions where fat consumption is higher they acquire a higher susceptibility for certain types of cancer. In addition, various epidemiological studies have implicated one or another fatty acid in the causation of one or another form of cancer. Other surveys, however, have suggested that the link between dietary fat and cancer is mediated through obesity. According to one view, some forms of cancer are diet sensitive whereas others are diet resistant; likewise, some forms of diet-sensitive cancers may be under the influence of certain nutritional factors, whereas others may be susceptible to different nutrients. Thus, on the basis of research in animals and from epidemiological data (2, 7, 47), a myriad of possible links, either promotion or protective, can be visualized among various dietary factors and different cancers (Figure 2). Currently, it is not possible to say with certainty that a cause-and-effect relationship exists in humans for any of these possible links. Moreover, recent epidemiological studies, if anything, have cast doubt on a specific causal relationship between dietary fat and cancer (52). Nonetheless, the possibility that high-fat intakes may predispose to some forms of cancer has engendered an enthusiasm for comparing high-fat versus low-fat (high-carbohydrate) diets in controlled clinical trials. Feasibility trials have been carried out, and at least one large clinical trial to test the hypothesis of benefit of very-low-fat diets is currently underway.

### *Osteoporosis*

Some concern has been expressed that very-low-fat diets may predispose individuals to osteoporosis. Such diets have been hypothetically linked to a negative

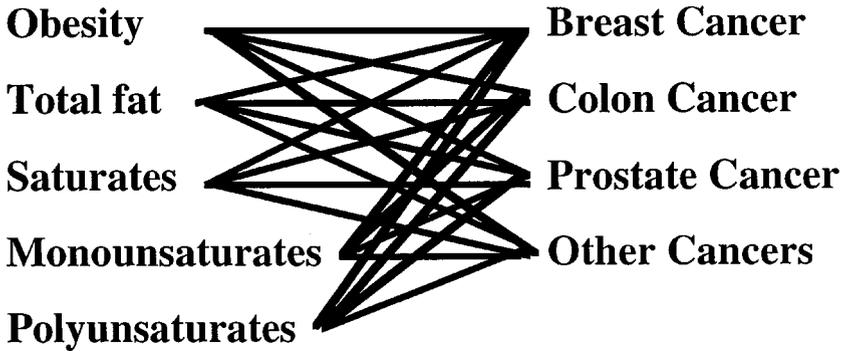


Figure 2 Potential links between dietary fat and fatty acids and development of various forms of cancer.

calcium balance in at least three ways. Very-low-fat diets, if not fortified, are typically low in calcium content. In addition, high intakes of fiber, which often accompany very-low-fat diets, may interfere with calcium absorption. Third, high intakes of carbohydrate may enhance renal excretion of calcium. Finally, if very-low-fat diets truly reduce body weight, lower weights could be a lesser stimulus for bone growth, whereas a low content of body fat may be accompanied by lower estrogen levels, which could promote osteoporosis. Each of these mechanisms deserves more investigation.

## MACRONUTRIENT RECOMMENDATIONS

Recommendations for dietary macronutrients must take into account not only relative proportions of total fat and total carbohydrate but also the composition of ingested fat and carbohydrate. Efforts to simplify dietary recommendations by specifying a percentage of total fat, for example without indicating the desirable fat composition, could be misleading if not counterproductive. Below, therefore, composition of fats and carbohydrates is addressed before turning to relative proportions of total fat and carbohydrate.

### *Dietary Fat Composition*

The actions of saturated fatty acids to raise serum LDL-cholesterol is well established. This effect has been documented in many human feeding studies as well as in animals. Moreover, a strong epidemiological association has been demonstrated between serum cholesterol levels and intake of saturated fatty acids in populations (28). The known link between elevated serum cholesterol and risk for CHD strongly implicates saturated fatty acids as atherogenic

fatty acids. To date, however, a large diet-heart trial has not been carried out to document whether reducing intakes of saturated fatty acids will decrease risk for CHD (40). Nonetheless, the circumstantial evidence implicating saturated fatty acids in atherogenesis is strong enough to warrant a recommendation for population-wide reduction in intakes of saturated fatty acids. Because *trans* fatty acids also raise LDL-cholesterol levels (36), they appropriately should be combined along with saturated fatty acids, and the sum could be designated cholesterol-raising fatty acids. Current recommendations (39, 41) of an upper limit of 10% of total energy as saturated fatty acids seems warranted, provided that they include *trans* fatty acids under this limit (Figure 1). If this recommendation is accepted, the population mean intake and recommended individual intakes of cholesterol-raising fatty acids should be about 7% of total calories.

### *Dietary Carbohydrate Composition*

There is a growing interest in the relative effects of simple sugars versus starches on metabolic processes. In several studies (21, 24, 32), evidence suggests that starch (complex digestible carbohydrate) has fewer adverse metabolic effects than do simple sugars, e.g. lesser postprandial glucose and insulin excursions (24), reduced hepatic fatty acid synthesis (22), and lower serum concentrations of triglyceride (32). Although this is an attractive hypothesis, it is not backed by a large body of solid research. Clearly, differences in metabolic responses to simple sugars and starches deserve more clinical investigation.

### *Percentage of Dietary Fat*

Most current recommendations of dietary fat favor a moderately low-fat diet (39, 41). Typical recommendations for total fat are “less than 30% fat”, “30% or less of fat”, or “up to 30% fat”. Although these recommendations are reasonable in that they try to balance to positive and negative sides of high-fat and very-low-fat diets, their meaning is ambiguous. Presumably these recommendations are meant to be targeted to individuals, although they are couched in terms of upper limits for populations (Figure 1). Thus, they do not provide meaningful guidance for individuals. Seemingly, a more appropriate recommendation for individuals would be for a percentage of fat intake to be approximately 30%. Presumably authoritative bodies recognize and fear that this recommendation will allow some individuals in the population to ingest more than 30% of energies as fat. This concern, however, can be lessened by accepting the concept that intakes of unsaturated fatty acids can be allowed to vary within the population provided that population intakes of cholesterol-raising fatty acids not exceed an upper limit of 10% of total energy intake. If cholesterol-raising fatty acids are reduced to recommended intakes, it is unlikely that total fat intakes will commonly exceed 30% of total energy. Should higher intakes occur in a

small portion of the population, little harm would result. In a word, the primary target for dietary intervention is a reduction in percentage intakes of animal fats and hydrogenated oils rich in *trans* fatty acids. If this change is made, there should be little concern about relative intakes of carbohydrates and unsaturated oils. On the other hand, in the absence of controlled trials that test for multiple endpoints of chronic disease, it seems prudent for individuals to avoid more extremes of carbohydrate or fat intake.

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